The average of all hourly movements of all seven men is shown below:

	First Series	Second Series	Average of Both
Normal	10.98	9.86	10.42
Hot water	8.35	8.51	8.43
Coffee	8.85	7.29	8.07

COMMENT

From these figures it is seen that in two series the average of movements per hour of seven men normally was 10.42. With hot water on retiring, it was 8.43. With cup of coffee on retiring, it was 8.07.

CHRONIC MENINGOCOCCEMIA*

REPORT OF CASES

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THE medical profession ordinarily thinks of meningococcus infections as meaning meningitis and naturally this phase of the disease, because of its gravity, has received the greatest amount of attention. The outstanding characteristics of this meningeal involvement are now universally recognized. The fact that at times meningococcus infection may result in chronic bacteremia with rather characteristic symptoms is not so well known, nor is it a matter of common knowledge that meningococcemia may cause lesions in any endothelial-lined structure of the body.

Netter[1] has called attention to the frequency with which chronic meningococcemia occurs. The reports in foreign literature suggest that either chronic bacteremia occurs more frequently in Europe or that the attention of their medical men has been directed to the fact that, meningococcus infection does not always mean meningitis, and that it may result in a general bacteremia, with or without involvement of any endothelial lined cavity, such as the synovial membrane, the endocardium, etc. In this country Warfield and Walker,[2] Morgan,[3] Cecil and Soper,[4] Krumbhaar and Cloud,[5] and others have reported several instances in which meningococcemia has produced ulcerative endocarditis without there being any evidence of meningeal infection.

Herrick,[6] who had ample opportunity to observe meningeal infections during the war, has shown that bacteremia precedes meningeal symptoms in the majority of cases. Other observers have corroborated these findings so it is not surprising to find an occasional case which never develops meningitis, and if developed, it does not occur until after the bacteremia has been present for a number of months.

The first proven case described of the meningococcus occurring in the blood stream was reported by Gwynn[7] in 1898. Two years elapsed before Salomon[8] reported a case of chronic meningococcemia. Since that time a number of cases have been reported in European literature. In this country relatively few cases have been reported. According to Dock,[9] only nine cases were reported up to 1911. Graves and Michelson[10] state that up to 1928 there were but fifteen proven cases of a chronic type of meningococcus bacteremia reported. Dock reports a number of cases which hardly fit in with the type of chronic meningococcemia here described because he included those cases "in which there was a febrile period of at least one week without meningeal symptoms and whose clinical course changed abruptly after meningitis supervened."

While it has been common knowledge that the fulminating type of case has resulted from an overwhelming blood infection, it has not been so well understood that the typical case of cerebrospinal meningitis is usually preceded by a bacteremia. Herrick and others in this country have shown that it is possible to secure positive blood cultures in about half these cases. Probably a larger percentage exists but the laboratory difficulties, together with the marked reduction of the organisms in the blood stream when the meninges become involved, make it exceedingly difficult to obtain positive cultures.

Warfield and Walker, Cecil and Soper, Rhoads,[11] Westenhoeffer,[12] Weichselbaum, and Ghon,[13] furnish evidence that endothelial lesions frequently occur. When the general practioner becomes conversant with this fact many cases of long and unexplained fever may be correctly diagnosed.

The earlier conceptions of meningococcus infection have geen decidedly modified in the last few years. We now feel that an acute meningococcus bacteremia precedes the usual type of cerebrospinal meningitis, the blood infection preceding the meningeal symptoms by a relatively short time. Positive blood cultures may be obtained early, depending upon the laboratory methods used.

Medical men are less familiar with acute fulminative meningococcus bacteremia which has been so well described by Herrick and others. This type represents an overwhelming infection with rapid onset, chills, marked prostration, a temperature which may be high or at times subnormal, pupuric eruption and the entire course lasting, as a rule, not over forty-eight hours and ending in death. At autopsy these cases may show no involvement of the meninges whatever, and the diagnosis is made from cultures.

Chronic meningococcemia represents rather a distinct clinical type of this infection. The onset is abrupt, there are chills followed by fever and at times there are vague joint pains and a rash. From the diagnostic standpoint the temperature curve is characteristic and important. It is frequently intermittent and is identical with the curve of the tertian or quartian type of malarial infection. Netter states that many patients have received treatment for a malarial infection because of the characteristic temperature curve.

* Read before the Southern California Medical Association at Long Beach, November 1, 1930.

One rather characteristic and interesting factor of this condition is the length of time the temperature may persist, several patients having been ill for a period of three or four months and one patient was reported as having had a temperature for seven months before meningeal symptoms occurred. Nearly all patients eventually develop meningitis if not diagnosed early and antimeningococcic serum administered.

Treatment by administration of polyvalent antimeningococcic serum is quite naturally most successful when given as early as possible. The amount of serum required varies decidedly in individual cases so far reported. The amount varies from 90 cubic centimeters; in one case reported 1000 cubic centimeters were given. After meningitis develops it would seem that the serum is not so efficient as one would expect, probably due to the general and prolonged bacteremia.

The two cases I am reporting seem, aside from their clinical interest, unique as they occurred in mother and son. Insofar as I am able to learn from literature, this disease has not been previously reported as occurring in two members of the same family.

SYMPTOMS

Usually the temperature is preceded by chills occurring on the second, third and fourth days, with quite definite regularity, without much prostration of the patient between the chills. In the second patient the chills were attributed to malaria, as he had had a definite malarial infection twenty years before, and lived in a section of the country where malaria is not unknown. When the intermittent type of temperature is accompanied by a leucocytosis, one questions the possibility of malarial infection. As the case progresses the temperature curve usually loses its intermittent character and becomes more constant, accompanied by chills and rather profuse sweats.

The joints are usually involved, the ankle joints and the knees more often than those of the upper extremities. Upon movement they are painful, at times swollen, but there is seldom any feeling of increased heat and as a rule they do not contain purulent material. The ankle joints of the second patient were much more painful than the knees; at times they were slightly swollen, never red and unless moved were not acutely painful. The sensation was described as a severe ache.

Skin eruptions are present in the majority of cases and are described as petechial, macular or nodular. The color varies from bluish to bright red. Some are painful, others not. They have been described in one case as dull red, nodular areas with white centers. It seems that the time and extent of the skin lesions are decidedly variable, occurring in some cases at the onset of the fever and continuing several weeks, in others occurring several weeks after the onset of the fever. In Case 2 the eruption was not noticed by the patient until after the third week of his illness, recurring at frequent intervals. They were from 1.0 to 1.5 cubic centimeters in diameter, distinctly nodular, hard and slightly tender to the touch, a dull red in color, fading gradually to a bluish discoloration which persisted for a week or more, and appearing in succeeding crops following the chills.

REPORT OF CASES

CASE 1.—R. M., female, age sixty-two, was admitted to St. Mary's Hospital September 1, 1926, having had a temperature for about twenty-four days.

Present Illness.—Pronounced and far advanced cardiorenal disease which had resulted in a cerebral thrombosis with complete right-sided hemiplegia and rather marked mental deterioration. Her condition required the constant attention of nurses because of mental confusion and incontinence.

About three weeks before her admission to the hospital it was noticed that she had a temperature which varied from 99.6 to 100 F. daily. During this time her pulse averaged from 70 to 104, with normal respiration. One week before admission to the hospital it was noticed that there was quite an increase in temperature, she was becoming exhausted, and the mental confusion seemed more pronounced.

The first two weeks she was in the hospital her temperature averaged from 100 to 101½ F. each day; highest usually in the evening. Agglutination tests for typhoid were negative; examination of the blood for malaria was negative. The leukocyte count averaged from 10,000 to 12,000, polymorphonuclear leukocytes from 65 to 76 per cent. The urine showed a moderate amount of albumin. The N. P. N. was at times as high as 100 milligrams. The creatinin varied from 4.5 to 6 milligrams. Blood cultures on September 14 and 17, 1926 were negative, but a culture taken on the 20th showed Gram-negative diplococci, agglutinating with antimeningococcic serum. It was deemed advisable to do a lumbar puncture. The spinal fluid was negative as regards cell count, globulin, etc. Within the next forty-eight hours 90 cubic centimeters of polyvalent antimeningococcic serum were administered intravenously. Her temperature subsided very promptly and she was discharged from the hospital on October 26, 1926. She lived until the following January, when she died of renal insufficiency without having a recurrence of her infection.

* * *

CASE 2.—S. M., male, age forty-three, was admitted to St. Mary's Hospital on May 23, 1929.

History of Present Illness.—Ten weeks prior to his illness he developed chills and fever of 103 F. which lasted about eight hours, following which he had no trouble for two days; then on the third day he had another chill and fever. This has occurred with definite regularity for about ten weeks. It was only after going to the hospital that he had a chill every day accompanied by a temperature of 99 F. in the mornings to 103 F. in the afternoon.

During the first four or five weeks of his illness he was well enough to supervise the work of forty or fifty employees for two succeeding days, but he always stayed home on the third day because he knew he would have a chill and feel very bad.

A few weeks after the onset of his trouble he noticed curious spots like flea-bites over his arms, hands, chest, and a few over his abdomen and the tibial surfaces of his legs. These lasted two or three days and then disappeared. About a week after the onset of his trouble he had aching in his joints, particularly those between the small bones of his hands, wrists, and ankles. The joints were never swollen or inflamed.

Three weeks after the onset of his trouble he consulted a local physician who thought he had malaria, inasmuch as there had been a history of malaria

twenty years before. Quinin was administered in large doses without results. Following this he consulted another physician who undoubtedly felt that he had some generalized infection, as he had a blood culture made with negative results. Following this he was advised to have six or seven infected teeth removed, which he did, without any improvement.

Because of the aching in the joints the patient thought that he had a rheumatic infection and on his own initiative he went to a hot springs, where he took a series of baths. These did not prove of any benefit to him. Shortly after this he was admitted to the hospital.

Past History.—Patient had had measles, chickenpox, whooping-cough; typhoid fever at the age of twenty, pneumonia one year later. Twenty years ago he had malaria, which lasted a few months. Tonsillectomy had been performed in childhood, but it is quite evident that the tonsils had only been clipped off. Double herniotomy had been performed eight years ago. In addition, he had a great deal of catarrh, as he expresses it, with a large amount of postnasal dripping.

Examination.—His appearance was that of a man in fair nutrition. He was quite weak, but not so weak as one would expect in an individual who had had a recurring temperature and chills for a period of ten weeks. Skin showed a decided eruption, which will be described later. The pupils were equal, regular, and reacted to light and accommodation. Fundi normal. The nose showed definite deflection of the septum to the right. Turbinates were enlarged. The left maxillary did not transilluminate clearly. The tonsils were very red and ragged. The anterior pillars were exceedingly red. Those teeth he had remaining were in fair repair. There was no enlargements of the thyroid, or other glandular enlargement.

Expansion was equal and regular on both sides. No change in the percussion notes and no unusual sounds were heard over the lung parenchyma.

The heart was slightly larger than normal. Blood pressure was 120.80. No murmurs.

There was no enlargement of the liver or spleen, and no areas of tenderness over the gall bladder or appendix. The rectum and prostate were normal. Reflexes were all present and equal. He had a maculopapular eruption, thirty or forty in number. These apparently came on in crops every few days, and usually after a severe chill. About one-half of them were distributed over his chest and abdomen, the others on the flexor surface of his arms and over the anterior surface of his legs below the knees. They varied in size from 1.0 to 1.5 centimeters in diameter, were decidedly raised, hard and somewhat painful. To the examining finger they were quite indurated and did not disappear upon pressure. The center of the eruption was not so red as the edge. They did not itch, nor was there any evidence of hemorrhage.

Blood Cultures.—After he was admitted to the hospital four blood cultures were made, all of which were negative. The blood was checked for Malta fever and typhoid with negative results.

The heart condition was of very great interest. At the time of his admission to the hospital no murmurs were detected although they were looked for. On June 1, one week later, a very low-toned systolic murmur was heard, loudest over the mitral area, and it was not transmitted. Within a few days this murmur became much more definite and a diagnosis of malignant endocarditis was made.

He insisted on leaving the hospital and five additional cultures were made. The eighth culture showed numerous small colonies of Gram-negative diplococci. Number 9 showed the same cultures as on the one on June 20, 1929. Subculture grown on blood serum showed agglutination with antimeningococci serum.

Diagnosis.—Meningococcemia.

Nose and throat cultures were made several times, and the fifth culture on June 20 showed the same type of organism that was found in the blood stream.

Treatment.—One hundred and eighty cubic centimeters of polyvalent antimeningococcic serum were given intramuscularly over a period of five days. Temperature remained normal after the fourth injection. An effort was made to give this serum intravenously, but with such a severe reaction, even after the ordinary procedure for desensitization was carried out that it was deemed advisable to give it intramuscularly. The length of his illness was fifteen weeks.

Subsequent History.—Following recovery he was not so well as before. He tired easily and had a great deal of aching in his feet, particularly around his heels.

Nasal cultures were made several times after his infection, with negative results.

Five months after his illness, he still had some nasal discharge, the culture from which was negative. One month later, nasal cultures were made on three different occasions on three different media, and they did not show any meningococci. At this time the heart was not enlarged and there was no evidence of a murmur.

March 3, 1930: Patient is apparently not so well as he was before his illness. He tires easily and his feet and ankles are quite tender and sore.

COMMENT

How the mother acquired the infection one may only speculate, certainly the few contacts, all of whom were well, limited the field. One might conclude that she was a carrier and thereby infected herself or acquired this infection from her son who was a chronic carrier. The second patient, the only one who had a nasal discharge, had a positive nasal culture two years and eight months later. It is reasonable to suppose that he was a chronic carrier of the organism and because of lowered resistance or increased virulence of the organism he developed a general septicemia. There are families who have a low resistance to certain organisms and this may serve as an explanation for the occurrence of two cases in this family of mother and son.

Whether or not there is an undifferentiated strain of the meningococcus which is more apt to produce septicemia and involvement of the endothelial structures has not as yet been determined.

The general condition of this second patient all through his illness was surprisingly good, much better than one would expect after chills and high fever had persisted for approximately four months.

The heart murmur was very interesting, developing as it did while he was in the hospital. This murmur was so decided that in conjunction with the chills and fever, joint pains and skin eruption, a diagnosis of malignant endocarditis was made. Within a short time, following the subsidence of temperature, the murmur disappeared. Whether or not the mitral endocardium was involved by the meningococci or the murmur

developed as a result of weakness of the myocardium from the continued toxemia, is a question of interest but one which cannot be decided.

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CEREBELLAR ATAXIAS

REPORT OF SIX CASES

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THE writers have encountered in the last few years six instructive cases of cerebellar ataxia which illustrate well how the various types of these cases merge and how extremely difficult the diagnosis may be in certain forms.

There is a basic symptomatology common to all cases whether hereditary or not, and whether beginning early in life or late. The various types of symptomatology known by the names of their discoverers are to the basic symptomatology as mountain peaks are to the earth—the same material is present, the difference being in the prominence of certain features.

REPORT OF CASES

CASE 1. (Illustrating the general, basic symptoms, not belonging in any particular type.) D. C., a man of thirty years, unmarried, was brought to us unable to walk because of inability to maintain his balance. There were no similar cases in his forebears. There was only one sibling, a married sister of thirty-two who was well and who had young children still healthy.

The history was obtained to the effect that the patient had unusual difficulty in learning to use his lower extremities when he first learned to walk at fifteen months. He had never walked well. At the age of six or seven years, speech difficulty became manifest in the form of jerky utterances, making pronunciation poor. At the age of ten or twelve he left school in the fourth grade because it was too great an effort for his parents to get him from his home to the schoolhouse. There was said to have been no progress of the disease for many years.

A neurological examination gave the following positive findings: Marked mental retardation, marked concentric contraction of the visual fields, bilateral paralysis of convergence with an irregular nystagmus. There was also limitation of movement of the eyes separately in every direction. In looking up, first one eye, then the other, overshot. At the fixation point a tremor was present. Both labyrinths were probably normal, but a central disturbance caused irregular responses which were elicited by very slight stimuli. The vocal cords were ataxic, causing sudden, unexpected changes in pitch. The speech was ataxic and slurring, at times indistinct, at times unintelligible. The head was held to the left.

There were involuntary, irregular movements of the pectoral muscles on the left. Respirations were irregular and jerky from ataxic movements of the chest and diaphragm. Adiadokokinesis was marked. The entire trunk was in a constant state of titubation whenever the patient attempted to sit or stand. Any tendency to fall was invariably overcorrected (hypermetria).

There were ataxic movements of the upper extremities, especially the left, but there was no rigidity. The vasti, lateralis of both thighs were rigid. Patellar reflexes were greatly increased, more on the left. Babinski and Chaddock signs were present bilaterally and also a Gordon reflex on the right. In attempting to walk, the patient went into a reeling, tottering, stumbling gait, which ended after not more than ten feet in a fall. His nasal bones had been broken several times in such falls. The spinal fluid was entirely negative.

There were no sensory disturbances, but quite a marked arteriosclerosis with elongation of the aorta as shown by fluoroscopic examination.

This patient showed the basic characteristics found in all types of cerebellar ataxia: First, a slow ataxia, manifested in cranial nerves, trunk, and extremities, giving rise to nystagmus, staccato speech, chorea-like movements of limbs and titubation, and second, signs of involvement of the pyramidal tract. He does not belong to any one type.

The pathology in these cases consists in a primary neuronic degeneration with secondary glial formations in the posterior columns of the cord; both direct and indirect cerebellar tracts and, of course, in Clark's column; the cerebellum and its direct connections; the ascending frontal convolution; and in the neurons of the retina.

The degeneration is first peripheral and a slow "dying back" toward the center occurs.

* * *

CASE 2. (Friedreich's ataxia.) C. S. A boy of seventeen years whose brother developed a similar condition at the age of thirteen and was helpless at the age of eighteen.

The patient had had measles at the age of two, influenza at ten, pertussis and chicken-pox at twelve, and typhoid at fifteen.

The trouble complained of had begun at the age of thirteen with awkwardness in getting about. (Usually the disturbance is not noticed until it has attained some degree of severity.) His strength and endurance remained good; he was still able to walk ten miles. His sleep was restless and somnambulism had occurred.

A neurological examination showed a dulled mentality with low intelligence. The right pupil was slightly larger than the left. A nystagmus was present on looking upward. Articulation was jerky and indistinct. There was a marked dorsal scoliosis. The deep reflexes in the arms were much diminished. The